Vol. 67, No. 10 Established 1901 October 1950

ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

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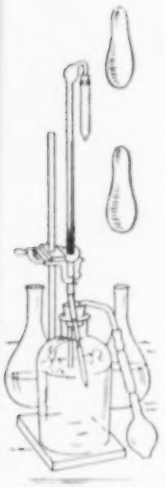

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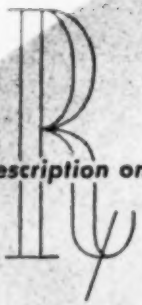

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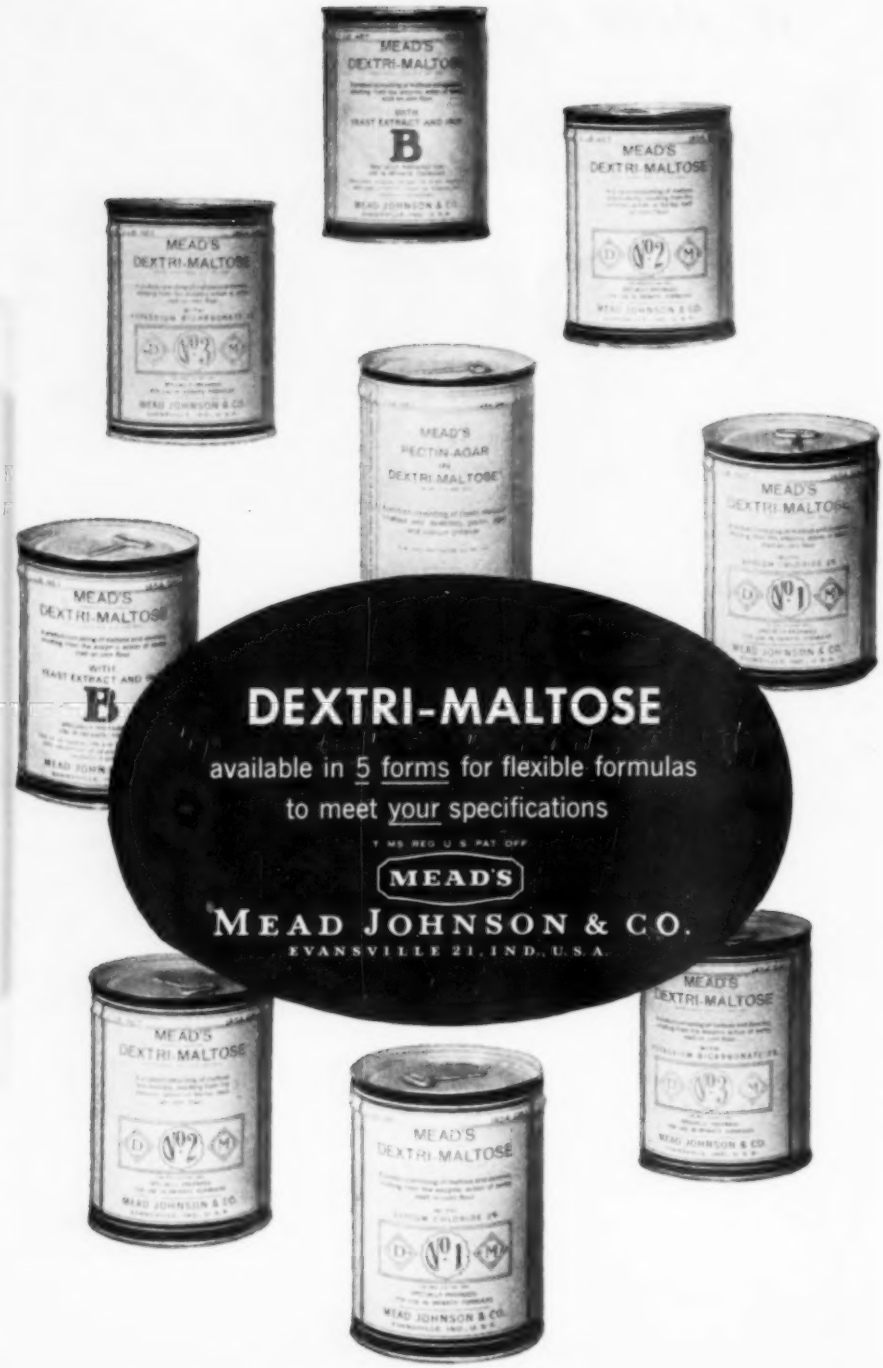
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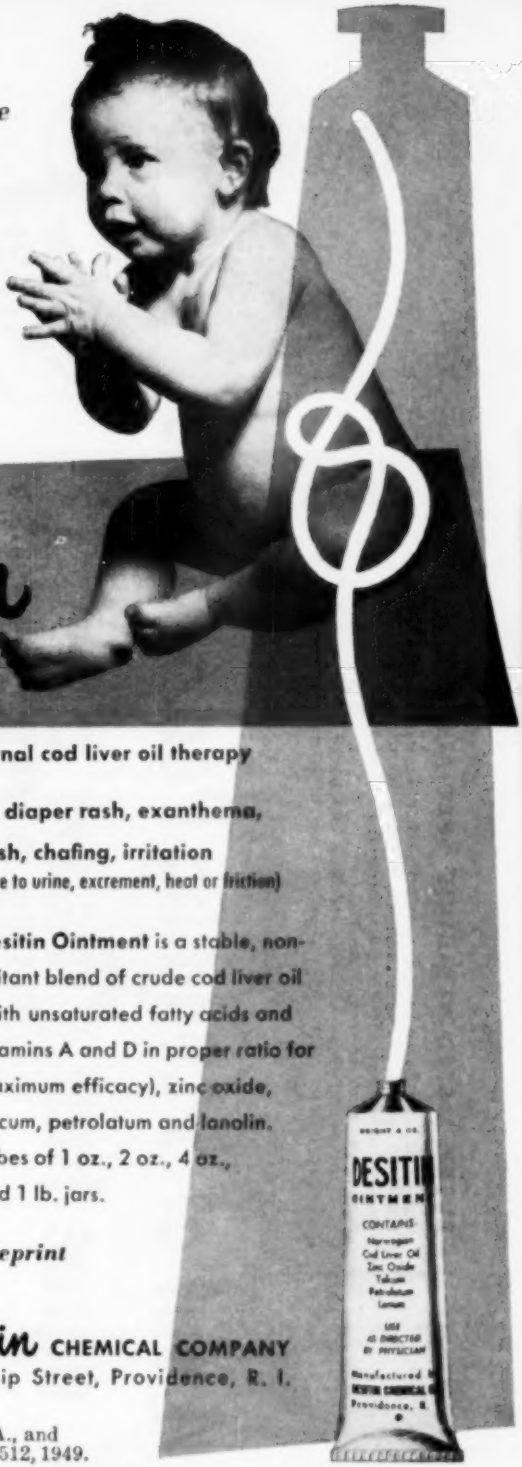

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1. Behrman, H. T., Combes, F. C., Bobroff, A., and Leviticus, R.: Industrial Med. & Surg. 18:512, 1949.

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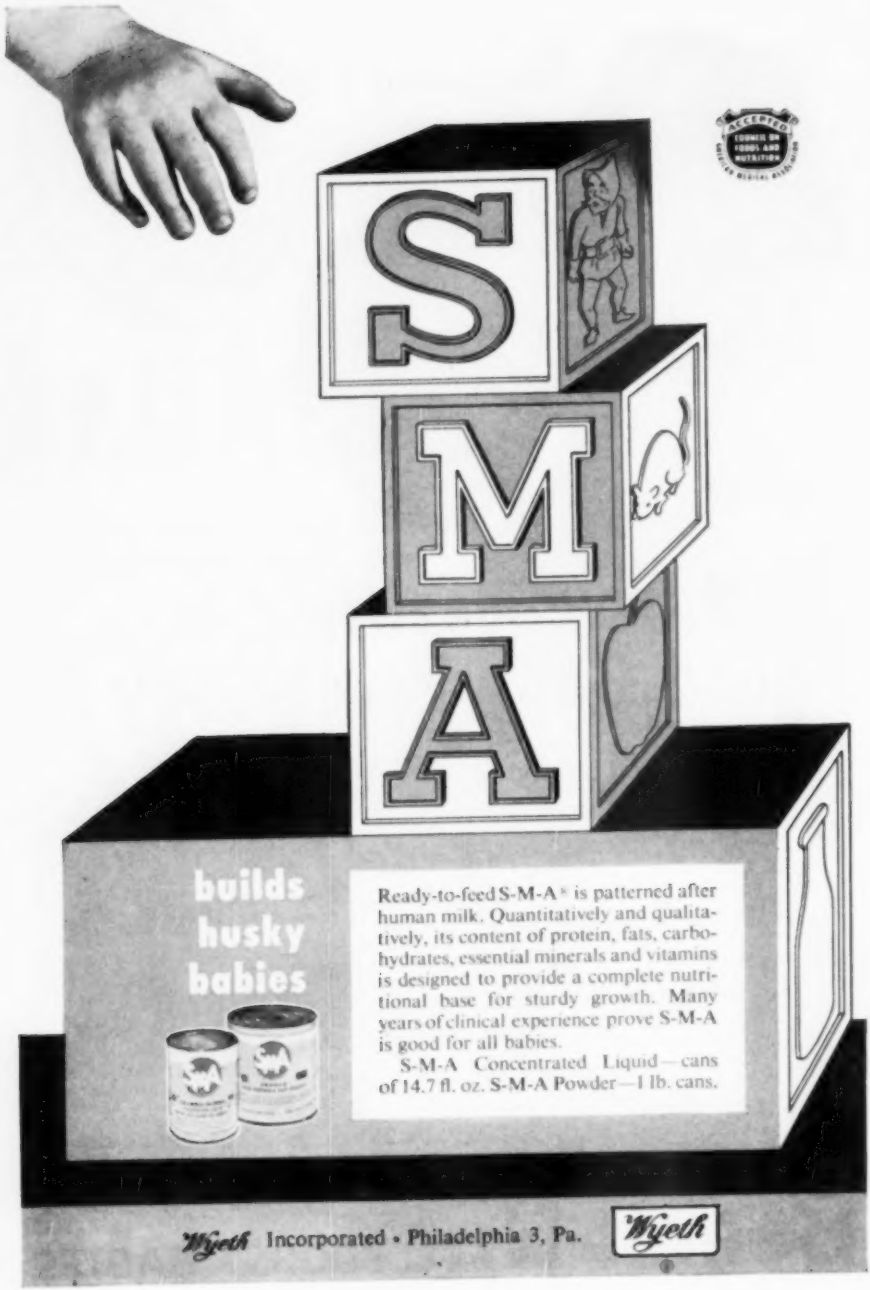
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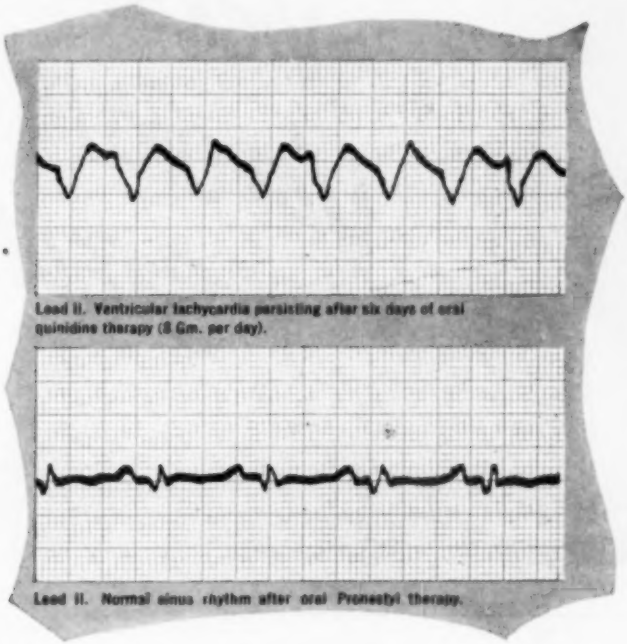

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NEUROBLASTOMA*

REPORT OF CASE

HOLLEY W. REED, M.D.

New York.

Neuroblastomas or sympathicoblastomas are the undifferentiated tumors of the sympathetic anlage (ganglion) and are usually found in the adrenal medulla or along the line of the paravertebral ganglions. The more differentiated types of sympathetic ganglion tumors are termed neurocytoma or ganglioneuroma. A great deal of confusion exists in the literature because of the many ways the tumors of the sympathetic ganglion present themselves to the clinician and pathologist. I use the term "neuroblastoma" in this report to denote the malignant member of the series of neoplasms arising from the sympathetic nervous system.

Stout[1] reports on 233 cases from the literature and adds ten of his own. He classifies the tumors of the sympathetic ganglion into three groups on the basis of malignacy. He reports that the commonest sources of origin are the sympathetic ganglia, which extend from the skull into the pelvis passing through the neck, posterior mediastinum, and the retroperitoneal regions including the adrenal medulla. He found the occurrence to be slightly higher in the females. The majority of neuroblastomas occur under the age of four, and they may be present at birth.

The majority of these tumors are circumscribed and at the onset often are encapsulated. The surface of these tumors is very vascular, as was observed in the case presented. The mass usually

*From the Department of Graduate Pediatrics, New York Medical College Flower and Fifth Avenue Hospitals.

does not invade but may displace the nearby organs or become bound up with important vital structures. The tumor spreads via the lymphatics and blood stream and late in the course of the disease it may invade the surrounding tissue. The tumor metastasizes most frequently to the orbit, liver, mediastinum and bone but may invade any area of the body. The bone metastases are very often bilaterally symmetrical.

The presenting complaint may be that the youngster has a black and blue eyelid, bulging eye, swollen glands or that he has bone pain. An abdominal mass may be the first clue that something is wrong. Blacklock[2] has reported that these tumors are associated with general symptoms, such as secondary anemia, frequently with a slight increase in the leukoytes, some of these being myelocytes. Fever is of an intermittent type. Anorexia and weight loss are usually present.

The following case is reported with the secondary anemia being stressed.

CASE REPORT

C. T., an 18-month-old negro male, was admitted to The Stamford Hospital, Stamford, Connecticut, for the first time on June 16, 1949, with the chief complaint of pains in his joints. The onset of his joint pains was two weeks prior to admission, and the pain had migrated from his left hip to his right shoulder. At one time there was some swelling of his right hip.

His mother's pregnancy was uncomplicated, and the birth was spontaneous and normal. His weight at birth was six pounds and upon discharge from the hospital, eight days later, six pounds two ounces. His growth and development were normal with only an occasional head cold. No history of familial diseases.

Physical Examination revealed a well-developed, well-nourished negro male in no accute distress, nor did he appear chronically ill. His temperature was 103° F. and he weighed 23½ pounds. The anterior fontanel was closed and examination of the eyes, ears, nose and throat revealed no abnormalities. The heart did not appear to be enlarged and no murmurs were heard. The chest was clear upon percussion and auscultation. There was no abdominal rigidity or distention. The liver and spleen were not palpable. No abdominal masses were felt at this time. No apparent joint

pain or tenderness. No limitation of motion of the extremities. Neurological examination was negative.

Laboratory Reports on admission revealed a red blood count of 3.66 million with a hemoglobin of 7.4 grams. White blood count was 11.3 thousand. White blood count differential revealed neutrophiles 69 per cent, of which 28 per cent were band forms and lymphocytes 31 per cent. Hematocrite 25 per cent and a corrected sedimentation rate (Cutler) of 30 m/hr. The red blood cells showed moderation hypochromia and moderate poikilocytosis and anisocytosis. Further blood counts are shown in Table 1. Urine was clear, pale yellow, with a specific gravity of 1.013; pH. 6; no sugar; slight trace of albumin. Red blood cells 1 to 2 per high power field; white blood cells 1 to 3 per high power field. There were no casts. The Wassermann and Mazzini were negative. Throat culture grew out beta hemolytic streptococci. The blood culture was sterile. X-ray examination of the chest was reported as showing no abnormalities of the thoracic cage. Both hilar regions were moderately accentuated. This accentuation extended into the second zone of the lung fields . Both costophrenic angles were clear, and the cardiac shadow appeared to be within normal limits. These findings were suggestive of a bilateral pneumonitis. On June 21, 1949, the heterophile and cold agglutination tests were negative. Complement fixation tests for typhoid O and H, typhoid A and B, proteus OX-19, tularenses and brucella were all negative. No sickling of the red cells.

Penicillin and acetylsalicylic acid brought the temperature down to normal and he was discharged on June 28, 1949, after having received 350 cc. of whole blood.

Final diagnosis was bronchopneumonia with a marked secondary anemia.

He was readmitted on July 2, 1949, with a fever of 101° to 102° F. of three days' duration. Since his discharge he had been very fussy, slept poorly and complained of soreness in his right leg. He vomited on the morning of admission.

Physical Examination revealed a very sick child who was listless when alone but cried when approached. The temperature was 102° F., pulse 200 and blood pressure 124/76. The ear drums were slightly injected and retracted. Abdominal examination revealed spasm of the rectus muscles, tenderness was equivocal and

TABLE 1.

	June 16	June 20	June 21	June 25	July 2	July 7	July 9	July 12	July 20	July 23	July 24	Aug. 5	Aug. 10	July 19 Father's Blood Count	July 19 Mother's Blood Count
Hemoglobin	7.4	6.4	..	8.4	7.9	6.1	..	8.0	..	..	6.3	..	7.5	14.5	12.8
R.B.C.	3.6	3.1	2.7	4.1	2.8	2.5	..	2.8	23	..	2.6	..	3	4.7	4.5
Hematocrit	25	20	..	..	26	..	..	..	..	..	20	32	..	42	40
M. C. V.	69	64	..	..	93	..	..	..	..	..	77	..	..	89	88
M. C. H.	21	20	..	..	28	..	..	..	..	..	24	..	..	31	28
W. B. C.	11.3	..	6.5	7.5	12.0	11.5	..	7.7	10.6	..	7	..	10.6	..	..
Neutrophils per cent	69	..	44	67	62	37	..	51	44	..	14	..	69	48	42
Bands	28	..	19	29	13	19	..	7	5	..	2	..	4	..	1
Lymphocytes	31	..	56	31	38	55	..	47	53	..	81	..	30	49	43
Monocytes	0	..	0	0	0	6	..	0	1	..	5	..	1	1	1
Eosinophils	0	..	0	0	0	2	..	2	2	..	0	..	0	2	14
Sed. rate	68	72	..	..	47	..	..	..	..	..	..	64	..	1	8
Corr. sed rate	30	23	..	..	18	..	..	..	..	..	..	22	..	..	..
Whole blood	..	..	350cc.	..	..	..	200cc.	..	..	300cc.	..	..	..	..	..

M. C. V. = Mean corpuscular volume. M. C. H. = Mean corpuscular hemoglobin. Moderate hypochromia, moderate macro- and micro-anisocytosis and moderate poikilocytosis were reported in all of the patient's smears. Red cell fragility was within normal limits. No sickling was noted in the patient or in either parents' smears.

there were no masses or distention at that time. There was no costovertebral angle tenderness. There was no lymphadenopathy and the neurological examination was negative. He cried when the left hip was moved and when the right thigh was palpated.

Blood counts during this admission are tabulated in Table 1. The urine showed a few coarse granular casts. The heterophile was 1:10. Coagulation time was less than ten minutes. Bleeding time was 23 minutes and 32 seconds. Clot retraction was normal. No reticulocytes in the peripheral blood. The platelet count was 160,000 per cu. mm. and the tourniquet test was negative after ten minutes. Icterus index was 5.7. Red cell fragility studies showed that hemolysis began at .48 and was not complete at .24. The control was .48 to .33. No sickling was ever noted. The calcium was 11.4 mg. per cent and the phosphorus 5.4 mg. per cent. Blood vitamin C level was 0.3 mg. per cent. Examination of the parents blood is reported in Table 1.

An x-ray on admission showed that the hilar accentuations presisted and were suggestive of an unresolved pneumonitis or atelectasis. On July 13 it was noted that the skin around the right eye was black and blue and that the eye was bulging. The eye became progressively more protuberant and by July 25 there was downward displacement of the globe. Two days later a rather soft mass was palpable at the upper and outer orbital margin. Ecchymosis appeared over this mass and this mass increased in size and extended back over the temporal bone. The proptosis soon became so marked that an eye shield was needed to protect the eye. Also on July 13 x-ray revealed evidence of some periosteal elevations along the shaft of either femur and of the left humerus. The diaphysis of the left femur was somewhat spongy in appearance and similar changes, though less marked, were noted on the opposite side. There was some flaring of the distal ends of the diaphysis of both femur and both tibiae.

On July 31 an abdominal mass was felt in the upper abdomen, slightly to the left of the midline. This mass was firm, non-tender and did not feel cystic. On August 1 intravenous pyelography showed the left kidney to be depressed downward and the right kidney was displaced to the right. Calices, pelvis and ureters were within normal limits. The abdominal mass increased in size and on August 4 an exploratory laparotomy was performed. A very

large retroperitoneal mass extended from the left gutter to the right gutter and up under the liver. It extended down under the kidneys and over the crest of the pelvis. The surface was covered with a great many dilated blood vessels. Examination of the stomach, small intestines and liver showed no evidence of metastases. A biopsy was taken and the abdomen closed. Shortly thereafter ecchymosis was noted around the left eye and soon proptosis was quite marked.

The patient became progressively worse and continued on a downhill course until he died on August 21. Autopsy was not permitted.

The blood work on the day of the operation showed total serum protein 6.85 mg. per cent, serum albumin 4.50 mg. per cent, serum globulin 2.35 mg. per cent. Prothrombin time was 16 seconds and the control was 15 seconds. Icterus 7. Bilirubin less than 1 mg. per cent. Blood urea nitrogen 12.7 mg. per cent.

The biopsy was stained with hemotoxylin and eosin and Nissl's stain (carbolthionin method) and was reported as ganglioneuroma, malignant.

COMMENTS

Cox[3] bases the diagnosis of neuroblastoma on (1) age of the patient; (2) the blood picture; (3) the presence of a palpable mass in the abdomen and (4) the observation of proptosis with orbital metastases. The case presented fits into the group that are under two years of age. The laboratory findings substantiate the existing belief that a severe secondary anemia often is associated with this tumor. Clinicians and pathologists agree that Wilms' tumor is the most frequent and that neuroblastoma is the second most frequent abdominal tumor of infancy and childhood. Abdominal palpation can not differentiate these two tumors or other retroperitoneal masses and, as with all solid tumors of the abdomen, absolute diagnosis can only be made after surgical removal and pathological studies have been made. The causes of proptosis are many, but when accompanied by an abdominal mass and bone changes in an infant or young child one is prompted to make a diagnosis of neuroblastoma.

Schmoyer[4] states that bone metastases may show (1) destruction of bone; (2) both endosteal destruction and proliferation and (3) those showing chiefly proliferation of the periosteum.

The use of the classification of these tumors into the Hutchinson type or the Pepper type has been discarded by the majority of writers, as overlapping of these two types has been reported by most observers. Suffice it to say that if this tumor were to be classified using these terms it would probably best fit under the Hutchinson type.

Surgical removal of the primary tumor followed by x-ray irradiation of the site and of the metastases is the treatment of neuroblastoma.

Although the prognosis of neuroblastoma is very poor, every effort should be made to effect a cure. Even in the face of metastases, treatment should be carried out, as Farber[5] has reported cases successfully treated by radiation after local metastases had occurred, although no cases have been reported that survived when bone metastases were present.[6] Farber has also pointed out that the tumor may undergo spontaneous hemorrhage and necrosis and disappear without any treatment other than biopsy. Cushing and Walbach[7] reported the spontaneous maturation of a malignant neuroblastoma into a benign ganglioneuroma.

SUMMARY

A case of neuroblastoma that had bone metastases and went on to a fatal outcome is reported.

A severe secondary anemia preceded the onset of symptoms and signs of the disease and continued throughout the course of the disease. This is in accord with the belief that an anemia often accompanies this disease and it is stressed in this report because it may be the earliest clue in the diagnosis of neuroblastoma.

Acknowledgment is made to Dr. Charles Murphy of Stamford, Connecticut, for allowing me to present this case.

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INFLUENCE OF BREAST FEEDING ON FACIAL DEVELOPMENT

FRANCIS M. POTTENGER, JR., M.D.
AND
BERNARD KROHN, M.D.
Monrovia, Calif.

Nursing in the first six months may control a person's facial contours for the rest of his life.[1] To determine this influence the authors measured an index of facial development in 327 cases. All age groups were represented in the series. It can be seen from these cases, as here presented, that breast feeding did benefit facial development markedly.

Good facial development is an essential stone in the foundation of good health. We see this best by considering the disorders that come from poor development. A face that is narrow in the frontal region contains crowded sinuses.[2] Another sign of poor development is a narrow dental arch. This brings on impactions, displaced teeth and malocclusion and agnathism. These defects interfere with the nutrition of the individual; they lead him to eat thoroughly cooked and macerated foods for the sake of easier mastication. But foods so prepared have lost important nutritive factors. For example, heating tomatoes in contact with air destroys their vitamin C content.[3] Cooking meats "well done" to make them tender denatures the unsaturated lipids which the body needs to make hormones.[4] A patient on such a diet will not ingest the proper building blocks to repair his body. His resistance to infection will be low; and he may develop degenerative diseases.[5] The first step to give a person the right nutrition is to make him able to eat sufficiently of the right food. This depends on the adequacy of his facial development; the strength of his muscles; the shape of his mastication bones.

The factors most apt to influence such development are (a) heredity,[6] (b) nutrition,[7] and (c) exercise.[8] To limit the influence of heredity in our study we selected all cases from the white race. To limit the influence of nutrition we chose all cases from a professional and business class that enjoyed a similar intake of the more sought after foods. This left nursing as the major variable factor responsible for the differences we were to observe. A child gets more exercise from nursing at the breast than on a bottle

because it requires more effort to extract the milk. The nursing infant works his jaw forward and backward, as well as up and down. The nursing infant works at a difficult extractive process and he stimulates the growth of his muscles of mastication, his mandible and his temporomandibular joint in so doing. However, we cannot ascribe all the differences noted to this factor. The quality of the mother's milk is in proportion to her health. Also, the composition of mother's milk is different from that of any cow-milk formula. Mother's milk contains the steroid first described by Dr. Wulzen, and it also contains phosphatases. The heat of pasteurization destroys the Wulzen factor and the phosphatase in cow's milk; but these are unaltered in mother's milk.... Steroids may be partly responsible for the better facial growth in breast-fed individuals.[7]

There is one more notable characteristic of the people studied. The majority of these patients came to us because of respiratory ailments. We would expect poor facial development more in these people than in the population at large. This was a group suffering from poor facial development.

The facial bones continue to grow well into adulthood.[9] Nevertheless, the growth of the first year leaves its mark which can be detected throughout life. We can demonstrate this in the bones to which the muscles of mastication attach.[10] The masseter is the chief muscle of mastication; and its bony origin in the malar prominence of the zygoma is easy to measure for size. The masseter pulls so it stimulates the malar prominence to grow both downward and laterally. A large malar prominence is of value, not only for aesthetic reasons and because it protects the eye, but also because it is the foundation of a powerful bite. The distance from the most lateral part of the right malar prominence to the corresponding part of the left malar prominence is called the bimalar distance; and it measures lateral malar growth. Just above the zygomatic bones are the frontal bones which come down lateral to the eyes. The distance from the right zygomatico-frontal suture to the left zygomatico-frontal suture is called the biorbital distance. The bones near the zygomatico-frontal suture enjoy the same nutritional and other circumstances as the malar prominences. This area near the suture is a definite landmark. Further, the bimalar and biorbital bony landmarks have almost no fat over them,

and are easy to measure. The difference between the two areas is important; the masseter muscle attaches to the malar prominence, but no muscle attaches to the area of the zygomatico-facial suture. So the biorbital distance is a control against which one can measure growth of the bimalar distance as influenced by the pull of the great muscle of mastication.

In each individual we measured the biorbital distance and the bimalar distance. The same physician made all the measurements. At a later hour a nurse took the history of how long the patient had been nursed. Fig. 1 shows how to measure the biorbital dis-

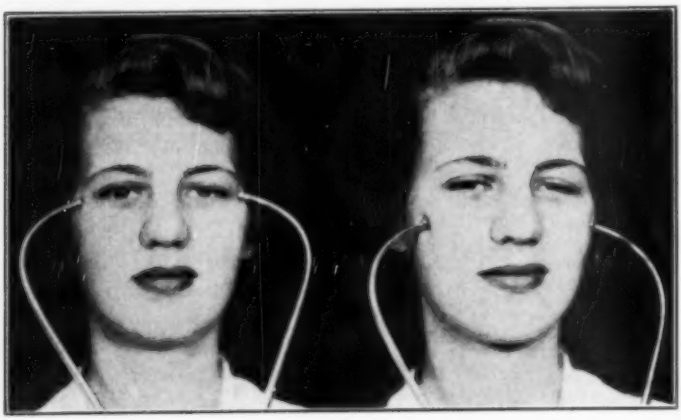

Fig. 1. Measuring the biorbital distance.

Fig. 2. Measuring the bimalar distance.

stance with a caliper; Fig. 2 shows how to measure the bimalar. Fig. 3 shows our findings for the whole series. Those persons who were not nursed at all showed the least malar growth, as found by comparing the bimalar and biorbital distances. Those nursed three months or less showed better malar growth; and those nursed more than three months showed the best malar growth of all. Fig. 4 contains our findings expressed as percentages. Here we see that the per cent of people who have well developed malar processes depends on the length of time they were nursed as infants. Thus, only 10.5 per cent of those not

nursed had a bimalar distance greater than their biorbital distance. Of those nursed three months and under, 23.6 per cent had a bimalar larger than their biorbital distance. Breast-feeding for more than three months caused the best facial develpoment; 57.2 per cent of these individuals had a bimalar larger than their biorbital distance.

FIG. 3. *Correlation of Nursing and Malar Growth*

Length of Time Nursed	Number of Cases	Biorbital Larger than Bimalar	Bimalar Larger than Biorbital	Biorbital and Bimalar Same Size
Not nursed.....	76	48	8	20
Nursed 3 months and under	55	34	13	8
Nursed more than 3 months	110	32	63	15
Nursed unknown length of time	86	11	61	14
Total number of cases..	327	..	..	..

If we analyze these cases by age groups, more becomes apparent:

1. The malar processes grow at least until the age of 25.

2. The bimalar distance is less than the biorbital distance in most children under 12, regardless of the amount of nursing.

FIG. 4. *Correlation of Nursing and Malar Growth In Percentages*

Length of Time Nursed	Number of Cases	Biorbital Larger than Bimalar	Bimalar Larger than Biorbital	Biorbital and Bimalar Same Size
Not nursed.....	76	63.2%	10.5%	26.4%
Nursed 3 months and under	55	61.8%	23.6%	14.5%
Nursed more than 3 months	110	29.1%	57.2%	13.6%
Nursed unknown length of time.....	86	12.8%	71.0%	16.3%
Total number of cases..	327	.....	.....	.....

3. Nursing increases the percentage of children under 12 who have bimalar distances larger than biorbital distances.

4. After 12 more children have well developed malar processes

FIG. 5. *Correlation of Nursing and Malar Growth Under 12 Years of Age*

Length of Time Nursed	Number of Cases A	Biorbital Larger than Bimalar	Bimalar Larger than Biorbital	Biorbital and Bimalar Same Size
Not nursed	46	36	1	9
Nursed 3 months and under	36	25	6	5
Nursed more than 3 months	22	14	4	4
Nursed unknown length of time	1	1	0	0
Total number of cases	105	..	..	..

FIG. 6. *Correlation of Nursing and Malar Growth Cases Under 12 Years of Age in Percentages*

Length of Time Nursed	Number of Cases	Biorbital Larger than Bimalar	Bimalar Larger than Biorbital	Biorbital and Bimalar Same Size
Not nursed	46	78.3%	2.2%	19.5%
Nursed 3 months and under	36	69.5%	16.7%	13.9%
Nursed more than 3 months	22	63.6%	18.2%	18.2%
Nursed unknown length of time	1	100 %	.....	.....
Total number of cases	105	.....	.....	.....

FIG. 7. *Correlation of Nursing and Malar Growth 12 Years to 25*

Length of Time Nursed	Number of Cases B	Biorbital Larger than Bimalar	Bimalar Larger than Biorbital	Biorbital and Bimalar Same Size
Not nursed	8	5	1	2
Nursed 3 months and under	12	9	2	1
Nursed more than 3 months	13	5	6	2
Nursed unknown length of time	7	2	3	2
Total number of cases	40	..	..	..

FIG. 8. *Correlation of Nursing and Malar Growth Cases 12 to 25 Years of Age in Percentages*

Length of Time Nursed	Number of Cases	Biorbital Larger than Bimalar	Bimalar Larger than Biorbital	Biorbital and Bimalar Same Size
Not nursed	8	62.5%	12.5%	25.0%
Nursed 3 months and under	12	75 %	16.7%	8.3%
Nursed more than 3 months	13	38.5%	4.2%	15.4%
Nursed unknown length of time	7	28.6%	43.0%	28.6%
Total number of cases	40	.....	.....	.....

Fig. 9. Correlation of Nursing and Malar Growth Cases Over 25 Years of Age

Length of Time Nursed	Number of Cases	Biorbital Larger than Bimalar	Bimalar Larger than Biorbital	Biorbital and Bimalar Same Size
Not nursed	22	7	6	9
Nursed 3 months and under	7	0	5	2
Nursed more than 3 months	75	13	53	9
Nursed unknown length of time	78	8	58	12
Total number of cases	182	..	..	..

FIG. 10. *Correlation of Nursing and Malar Growth Cases Over 25 Years of Age in Percentages*

Length of Time Nursed	Number of Cases	Biorbital Larger than Bimalar	Bimalar Larger than Biorbital	Biorbital and Bimalar Same Size
Not nursed	22	31.8%	27.3%	40.9%
Nursed 3 months and under	7	.....	71.4%	28.6%
Nursed more than 3 months	75	17.3%	70.7%	12.0%
Nursed unknown length of time	78	10.2%	74.4%	15.4%
Total number of cases	182	.....	.....	.....

than have poorly developed ones. This occurs only if they have been nursed more than 3 months. If they have been nursed less than 3 months, the great majority will have poorly developed zygomatic bones.

5. Most people over 25 have fairly well developed malar processes, regardless of whether or not they were nursed as infants. But good malar development is still more common among the ones who were well nursed.

The following statistical analysis supports the five conclusions above: Figs. 5, 7 and 9 contain the findings on our 327 cases grouped according to ages. Figs. 6, 8 and 10 are this material expressed in percentage terms.

1. In Figs. 6, 8 and 10 the percentage of cases having a larger bimalar than biorbital increases with age in each category. This shows that the malar processes do continue to develop into adulthood.

2. In Fig. 6, 63.6 per cent had larger biorbital distances than bimalar distances, even after being adequately nursed. These children were too young to have optimally proportioned faces.

3. But even among these youngsters, 18.2 per cent of those nursed over 3 months had a larger bimalar than biorbital, compared to only 16.7 per cent among those nursed 3 months or under, and 2.2 per cent among those not nursed at all.

4. In Fig. 8 we have the group between 12 and 25. In those nursed more than 3 months, 46.2 per cent had a larger bimalar than biorbital, compared to only 38.5 per cent who had a larger biorbital than bimalar. In those nursed under 3 months only 16.7 per cent had a larger bimalar than biorbital. In those not nursed at all only 12.5 per cent had a larger bimalar.

5. In the cases over 25 years of age shown in Fig. 10, we find that over 70 per cent of those who were nursed have a larger bimalar distance than their biorbital. Among those not nursed, only 27.3 per cent had a larger bimalar.

CONCLUSIONS

The findings in our 327 cases indicate that it is advisable to nurse a child at least 3 months, and preferably 6 months. This will stimulate optimal malar development. We have also observed that patients who were well nursed had better developed dental

arches, palates and other facial structures than patients who were not nursed. A series measuring the growth of these and other facial structures would illuminate this subject further. Another point is worth considering: some mothers could not supply milk because they were in a poor state of nutrition. This, plus lack of the exercise involved in breast-feeding, foredoomed the children to poor development. To complete our program we should aim to get every pregnant woman in condition to nurse her infant.

SUMMARY

1. Development of the zygomatic bones is important to the face for aesthetic, protective and functional reasons.

2. Development of the malar prominences of the zygomatic bones was used as an index of facial development.

3. It was found that breast-feeding improved the growth of the malar processes.

4. Malar processes grew until the age of 25, at least. But the people who were nursed got off to a head start; and the others never caught up to them.

5. It is advisable to get all mothers into a good state of nutrition and to have them nurse their infants.

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609 North Canyon Boulevard.

POISONING AS THE CAUSE OF POLIOMYELITIS

RALPH R. SCOBEY, M.D.
Syracuse, N. Y.

(Continued from the September issue)

PARALYSIS FROM MIASM *(continued)*

Pascalis[127] (1824) speaks about miasm as a cause of intermittent fevers. He says: "That such exposure proves generally unhealthful, it is readily granted, and they cause rheumatism, palsies, cramp, and a variety of other complaints."

Conventry[128] (1824), writing on endemic fever in the Genesee or Lake Country of New York State, says that it begins with the most violent arterial action; yet ends in the most marked nervous debility. He states that Hippocrates, many centuries ago, described the disease under the appelation "causus."

"During the years mentioned (1792-1795)," he says, "I can truly say that few days occurred, from the middle or latter part of July to the setting in of the frost, about the middle of October, that did not afford me the opportunity of visiting a patient with endemic fever. Having for forty years observed the species of fever spoken of, always accompanied by, and never independent of vegetable and animal decomposition, that it was dependent on, or connected with that process. The deleterious effects of vegetable and animal decomposition on the human body are established by innumerable facts with which the records of medicine abound. . . . Quarantines have been faithfully tried but are found wanting," he says.

Willoughby,[129] in a letter to Dr. Conventry in 1824, describing endemic fever in Herkimer County, New York State, says: "There was one symptom, and which might be considered as rather premonitory than otherwise, usually present this year (1803) which I have repeatedly noticed and subsequently, and which I do not recollect of seeing noticed by any writer. It was a certain diagnosis of fever, and by it I have been enabled to predict with certainty of fever before the patient was in the least aware of its danger, or hardly considered himself indisposed. This was a stiffness and aching sensation of the muscles in the back part of the neck. . . . I am certain that the decomposition of dead organized

matter, whether it be animal or vegetable, or both combined, when acted upon by heat and moisture, produces a poison (miasm if you please) which is the common exciting cause of itself over the whole system, deranging its functions."

Coventry[130] (1825) points out that the parts of the body most frequently affected in endemic fever are the head and the stomach. He speaks about perishable materials in basins, slips, docks, ponds, marshes and stagnant pools. "The cause is the same; and will be found accompanied by vegetable or vegetable and animal putrefaction. To produce and induce this process, heat and moisture are the principal agents; hence it prevails in those countries where these agents operate powerfully, and find pabulum to act on. The effects are varied by the idiosyncracy of the individual, and the intensity of the agent, as to induce respectable physicians to make distinctions where there is no difference. . . . The idea of contagion and importation is now so completely abandoned, that it would be an act of supererogation to dwell on the subject," Conventry says.

Fountain[131] (1827), writing on Intermittent Fever, points out that at no period within the space of fifteen years has intermittent fever prevailed so generally throughout the State of New York as during the last two years. "There is but one set of exciting causes, and this included all miasmic exhalations from marshes, soils, etc. The cause of intermittent fever is confessedly an invisible substance, the product of the play of chemical affinities among the materials of marshes, soils, etc., and all agents acting primarily on the nervous system; it follows of course, that the first evidence of disorder are manifestations of this tissue, acting under the combined influence of the morbific agent, and the inherent tendency of the living power to maintain its own movement. This complicated and confused respondence of the nervous system continues longer or shorter according to the feebleness of the morbific agent and the constitution of the living fiber. . . . I have known it induced by fright and by falling from a horse, and in these cases it came on instantly and pursued an uninterrupted course with its usual type. Besides it has been known to be a local disease affecting one arm only."

Fountain records the case of Eli Ganong of Putnam County, New York, who had a mild remittent fever. On the sixth day

he found that he could not move his hands or feet; and examination proved that they were paralyzed. The paralysis gradually progressed up his legs and arms, and on the fourteenth day they reached the muscles of respiration, and death ensued from their insubordination to the will. Sensibility of his mental faculties were unaffected.

Fountain points out how the various manifestations that he observed could result from arsenic but are frequently the effects of intermittent fever. "Yet every instance of the kind," he says, "occurring under the influence of arsenic would be attributed to this article." He speaks of a case of remittent fever which was denominated typhus fever, which he points out was a very convenient subterfuge for indolent practitioners.

Seymour[132] (1828, 1839) endeavored to show that poisons received into the circulation from the atmosphere act as mineral and vegetable poisons do on definite structures of the body. He points out that epidemic fever, according to the generally received opinion, originates from a poison arising from the marshes. This view, he says, has occupied the attention of physicians and men of observation from the earliest ages. "A correlation with the other poisonings has been pointed out for over a half century," he states, "It is by no means necessary, nor is it probable that miasm generated in the manner related should produce the same poison, or consequently affect the same texture uniformly; and thus we find epidemic fevers in which the poison appears to act directly on the brain, and various effusions in, or lesions of this viscus, are discovered after death."

Chambers[133] (1828) points out that remittent fever prevails in India and China, and other hot climates, in the unhealthiest season, viz., in August and September. The remittent fevers of England, he says, although differing from that of the hot climates, are the same disease in essence. This important observation appears to correlate the observations of Boerhaave[134] (1761), Shaw[135] (1823), and Goodeve[136] (1879) who pointed out that illnesses exist in India and China that result in paralysis.

Chambers emphasizes that this fever is not contagious; that physicians do not contract it from patients; and that other attendants do not contract it. He points out that there is no record of the disease spreading to other patients in a hospital.

Hawkins[137] (1829), England, speaks of remittent fever sometimes assuming an intermittent form, called arachnitis. Arachnitis of the base of the brain, he points out, produces rigidity of the muscles of the neck, whereas spinal arachnitis causes a general stiffness and curvature of the whole spine with locomotor powers affected. "It has happened," he says, "that the symptoms of other disorders which have been complicated with arachnitis have become the most prominent and have attracted the chief attention."

Mac Culloch[138] (1830) states: "Paralytic affections make their appearance also as a substitute for, or as a symptom in, both acute and chronic intermittents. Sometimes they occur in the course of it, and sometimes the original disease terminates in them."

Turner[139] (1832) describes the dry belly-ache of the West Indies, which he points out is of common occurrence there, proving fatal in many instances, and in others followed by paralysis. He felt that it was caused by lead poisoning. Musgrave[140] (1833), in a reply to Dr. Turner, denies that this disease is the result of lead poisoning. He felt that it was of malarious origin and that he had occasionally seen it in epidemic proportions. He points out that the disease occasionally occurs in town but is regarded as belonging to the country.

The modern virus theory, employed to explain the spread of poliomyelitis today, is reminiscent of animalcular theory employed to explain the miasmic diseases of the past. Caldwell[141] (1831) speaks of advocates of this theory as follows: "In their opinion putrefaction of marshes produced myriads of animalculae too minute to be detected by our senses or to become cognizable in any way, except by their effects on larger forms of living matter. These monads of life, as small and as numerous as particles of air, make their way into the human body by the pores of the skin, or in some other manner, mingle with the fluids, pierce and poison the solids, and spread corruption through the whole, then propagating their like with powerful fecundity, they issue from the bodies of the sick to invade those of the well, and thus the disease is rendered contagious. This hypothesis of animalcular contagion, however wild and irrational it may be deemed, has its advocates even now." Caldwell points out that the work of Lancisi, in 1717, was instrumental in discarding the animalcular

theory. Thus a "virus" theory became obsolete 233 years ago which was employed before then by some to explain illnesses that frequently resulted in paralysis.

Whereas the remittent and intermittent fevers of adults were generally attributed to miasm, those of children began to be associated by early writers with other factors, such as food, teething, foul bowels, worms, etc., as well as miasm.

Ashburner[12] (1834) says of infantile remittent fever: "That the train of events constituting the infantile remittent or developmental fever is but a part of that which leads to hydrocephalus acutus, that the one disease is a lower grade of the other is a proposition which seems to my mind too clear to require additional illustration."

Ramsbottom[13] (1835), England, points out that infantile remittent fever is most commonly occasioned in the older child by his being indulged in improper or unusual articles of diet with irritation of the teeth as an exciting cause.

Michael Underwood,[14] England, is considered to be the first to describe the disease that is now called Infantile Paralysis or Poliomyelitis. This supposition is based on the description of a disease entity which appears in the 2nd Edition of his book on diseases of children published in 1789. However, in the 9th American Edition of this same book, published in 1841, two years after Heine's reports[15] of Infantile Paralysis, this same disease would appear to have been relatively frequent in those days since under the heading of Paralysis or Palsy, for example, it is stated: "Paralysis is a more common disorder in infants and young children than writers seem to have imagined; it is confined to no age and attacks children in very different degrees, in the manner it does adult persons. It accordingly, sometimes, seizes the upper, and sometimes the lower extremities; in some cases, it takes away entire use of the limbs it has attacked, and in others, only weakens them. . . . Besides teething infants, whom it has said to attack, I have seen it in others who are older, and the finest of children, and it is often generally attended with a foul state of the bowels."

Armstrong's description of remittent fever appears to be pertinent to these cases of paralysis. He says: "The infantile fever, the infantile remittent, the biliary remittent of infants, or as it is sometimes, but improperly, called, the worm fever, generally

arises from indigestion, occasioned by food of improper quality, or too abundant in quantity. . . . It is a disease so far 'void of danger', that it frequently excites the greatest apprehensions, and sometimes proves fatal, or lays the foundation for other disorders, that are ultimately to end fatally."

Boling[146] (1846), writing on remittent fever says: "Heat, moisture, and the decay and decomposition of rich and luxuriant vegetation are the three principal causes to which its production is attributed, and certainly it gives manifestations of its presence under the existence of this combination more frequently than under any other." He speaks of a host of painful and anomalous neurological affections resulting from these agencies. "Remittent fever," he says, "has been divided into many varieties, dependent frequently upon the fancy of the writer, and having no real separate existence in nature: the different varieties passing by imperceptible shades into each other. The predisposition having already been engendered by the poisonous agent, there is no symptom more strikingly characteristic of remittent fever than excessive irritability of the stomach." He speaks about the bowels often being costive.

At about this time, Emerson[147] (1848) reported on the vital statistics of Philadelphia for the decennial period from 1830 to 1840. He points out that this city, with a population of 226,693, had a formidable number of deaths from diseases of the nervous system. "Convulsions claim, as usual," he says, "the largest proportion of victims recorded under a separate head, the whole amount during the decennial period being 2,985. Hydrocephalus is charged with 1,854 deaths, cephalitis 1,095, delirium tremens 826, apoplexy 704, palsy 471, epilepsy 108, insanity 104, tetanus 51, and chorea 10.

Stone[148] (1846), writing on miasmic diseases in the Western United States, asks and answers, "What then is a physician to treat, when called to the bedside of a sick patient? He has to treat a case of poison?"

Lawton[149] (1848) states that there is formed by decomposition a something which we call malaria, long known by its effect, being followed by a train of diseases peculiar to themselves, and known to be produced by no other morbific agent.

Kennedy[150] (1850), writing on the paralysis in early life, alludes

to those forms of the disease which occur subsequent to the fevers. "No matter what type they may assume," he says, "though it is most probably most usual to meet them after gastric and infantile remittent fevers, such cases are of tolerably frequent occurrence, and may be almost set down as forming a distinct group of themselves."

Churchill[151] (1850), in his book on diseases of infants and children, describes infantile remittent fever as follows: "It is a species of fever to which children of one year old to ten or twelve are very liable, characterized by one or more remissions, by pain of the belly, and sometimes, also, of the head, and by an unusual state of the alvine discharges. Such attacks are always attributed to worms by the older writers. Dr. Joy noted tenderness and intolerance of pressure in the upper part of the spine."

Churchill points out that there is irritation of the cerebro-spinal system in consequence of continued irritation of the intestines. Cold and teething are two principal causes of remittent fever as well as irregular or unwholesome food and it may occur in epidemics, he says. Sims, he points out, has described epidemics of infantile remittent fever which occur at the same time with the prevalence of a low nervous fever among adults. "The great difficulty in the diagnosis of remittent fever," Churchill says, "is from meningitis; and, no doubt, it is often a very difficult point."

Sawyer[152] (1858), writing "On the Vital Statistics and Causes of Mortality in San Francisco," states: "Cases of paralysis of every degree are of common occurrence here. In some instances it apparently has miasmic origin."

That infantile paralysis was of frequent occurrence at that time, and before, is indicated by Taylor[153] (1866) in his book on Infantile Paralysis, when he says: "There is sufficient reason for the fact that paralysis is of common occurrence in early life." The general practitioner apparently took these cases for granted and apparently designated them by various names. As a clinical entity infantile paralysis originated from specialists, rather than from practitioners, as the early reports of this disease clearly establish. The meagreness of the literature on the subject of infantile paralysis is thus explained by Taylor, an orthopedic specialist of New York Orthopedic Institute. "Only when some enthusiastic 'specialist' calls them from their hiding place—few in a given locality

but many in community—is there sufficient opportunity for that consecutive and multiplied observation necessary for establishing facts and making deductions." It is not surprising, therefore, that the early reports of infantile paralysis, as a disease entity and the end result of an illness not always ending in paralysis, should have originated from such orthopedic specialists as Underwood[154] (1789), Joerg[155] (1819), Stromeyer[156] (1833), Badham[157] (1834), Heine[158] (1838), and others; and later from neurologists after neurology also became a specialty. The increasingly frequent reports of operations for torticollis, strabismus, clubfoot, and curvature of the spine, as well as the establishment of orthopedic institutions in the first half of the 19th century, is of great significance.

Lanzi[159] (1876) made microscopic studies of the water of marshes, and as a result he considered the algae, passing into a state of putrefaction in the water, to be responsible for malarial fevers.

Lincoln[160] (1887), writing on, "The Climate and Health of Savannah" which covers a ten year period, states: "On the whole, the southern city presents a very high mortality from nervous diseases and miasmic fevers, and a rather high rate from digestive disorders."

Wood[161] (1889) states that a great many deaths, registered in South Australia during the period 1879-1880, were registered under the heading of paralysis. During these same years, he points out, malarial fever was very prevalent.

Bondurant[162] (1900) describes an epidemic of poliomyelitis in Green County, Alabama, in which there was much malaria at the same time. Included in this report are "some adult cases thought to be endemic of malarial neuritis." There was no evidence of poliomyelitis in the rest of the state.

The term "malarial fevers," which included a medley of diseases, was retained for some time after the discovery of the malarial parasite by Lavaran in 1880. Thus, it is to be noted that reports on the mortality in New York City during July 1907,[163] at a time when a severe epidemic of poliomyelitis was prevalent, recorded an augmented mortality from malarial fevers. There was also a rapid increase in the deaths from diarrheal diseases in infants and young children.

THE POLIOMYELITIS VIRUS: AN ENDOGENOUS PRODUCT

Since the claim of discovery of the so-called virus of poliomyelitis by Landsteiner[164] in 1908, every attempt made to show that this so-called virus enters the human body, under natural circumstances, to cause the disease has failed. It cannot be denied, of course, that a substance obtained from poliomyelitis victims, from sewage, flies, etc., commonly called a virus, exists and that it will produce a disease in experimental animals characterized by paralysis with lesions in the anterior horn cells of the spinal cord. Conclusive proof, however, that this so-called virus ever enters the human body, under natural circumstances, to cause the disease is conspicuously and definitely lacking.

There is a considerable amount of controversy concerning this portal of entry of the so-called virus into the human body. Thus, we find in the medical literature during the past decade such statements as the following: Aycock and Eaton[165] (1925): "Our present knowledge permits only speculation concerning the mode of infection"; Lumsden[166] (1938): "The views as to the portal of entry and communicability rest fundamentally and entirely on findings from experiments on monkeys. To some of us it does not appear to square with the facts obtained by epidemiological studies of the disease among human beings. It seems too elastic, too restful. On epidemiological grounds alone, it appears conceivable that poliomyelitis is not caused by a living microorganism or a virus, but by a toxin."; Neal[167] (1939): "The portal of entry of the virus is still a hotly debated question."; Sabin[168] (1939): "We possess no knowledge of the actual portal of entry into the human body"; McKhann[169] (1940): "The portal of entry of the virus has not been definitely established"; Smith et al.[170] (1945): "It is well-known that during and for some time after an epidemic the virus can be isolated from sewage, from outside toilets, occasionally from polluted and stagnant water, and in a few instances from flies which have been caught in nearby areas. Under some of the conditions the virus may remain active for several months. Theoretically, such non-human reservoirs would be expected to play an important role in the initiation of an epidemic and in the spread of the disease from person to person. Yet, in spite of the comparative ease with which polio may be experimentally transferred to monkeys from these reservoirs, it

has not been established that the virus in such form gives rise to the human disease. The possibility exists, but direct proof is still lacking." A perusal of contemporary reports on poliomyelitis (1950) shows similar statements, controversies, and speculations.

Although the literature for 35 years showed no record of "infection" of laboratory workers by the so-called virus of poliomyelitis, the accidental inoculation of an occasional laboratory worker during recent years has given renewed impetus to the hypothesis that a virus enters the human body to cause poliomyelitis. However, one could naturally expect if the so-called virus of poliomyelitis, whatever its nature, is accidently or intentionally injected into a human that it would obviously cause a disease similar to that produced in experimental animals by inoculation It must be admited, however, that this is an abnormal manner of acquiring a disease.

Experimental evidence, on the other hand, appears conclusively to have shown that the so-called virus of poliomyelitis does not enter the human body, under natural circumstances, to cause the disease. Experiments, intended to show that a virus enters the human body in a natural manner through the respiratory and gastro-intestinal tracts and the skin have been uniformly unsuccessful.

The experiments of Schultz and Gebhardt[171, 172] (1937) can be considered as almost conclusive proof that the so-called virus of poliomyelitis does not enter the human body by way of the respiratory tract. These workers found that the application of zinc sulfate as an astringent barrier when applied to the nasal mucosa of experimental animals prevented the passage of the so-called virus in the body. As a result of these experiments, zinc sulfate was applied to the nostrils of thousands of children in Toronto during an epidemic of poliomyelitis in 1937.[172] The results of these experiments, when analyzed, proved that the zinc sulfate applications had been ineffective in protecting children from poliomyelitis during that epidemic when compared with the results from the untreated control group.

A number of workers have likewise been unable to "infect" experimental animals by way of the gastro-intestinal tract. Howe and Bodian[174] (1939) had uniformly unsuccessful results in showing that the so-called virus enters the body at this point, as did

also Faber et al.[175, 176] (1943; 1948). These workers found that the gastro-intestinal mucosa in fact resisted the passage of the so-called virus of poliomyelitis. This fact was shown in their attempts to "infect" 67 monkeys by means of capsules heavily laden with the so-called virus. It was recently reported also that attempts to "infect" monkeys with the so-called Coxsacki virus by way of the gastro-intestinal tract were also uniformly unsuccessful.[177]

All of these unsuccessful attempts to "infect" animals indicate that although a substance, considered to be a virus, is found in the gastro-intestinal tract of cases of poliomyelitis, and subsequently in sewage, yet such evidence is in no way an indication that a similar substance ever entered the gastro-intestinal tract. One is forced to the conclusion that the gastro-intestinal tract contains, in cases of human poliomyelitis, and perhaps in the gastro-intestinal tract in the course of other diseases as well, a chemical substance which will produce a disease characterized by paralysis when *injected* into experimental animals.

Proof that a virus does not enter the human body through the skin, which it could almost certainly not do during epidemics of poliomyelitis except by an insect bite, is indicated by the fact that epidemics of this disease appear in many instances before the appearance of insects and continuance after insects have disappeared. The ineffectiveness of DDT and other insecticides in checking epidemics of poliomyelitis is further proof of the improbability of the skin as a portal of entry.

The portals of entry of a virus into the human body, under natural circumstances, to cause poliomyelitis has not only not been established, but the communicability of this disease after its supposed entry, has not been proven.

The theory that poliomyelitis is communicable has never been able to account for such anomalous and contradictory facts as the victimization of individuals who have had no contact with active cases; the non-communicability to doctors, nurses, and ward attendants; the absence of communicability to patients in hospitals and to individuals in communities when quarantines are not established; the rarity of multiple cases in the same family even where a child with poliomyelitis is known to have slept with another child; its greater incidence in small communities than in

large cities where crowding exists and where, consequently, poliomyelitis should extort a staggering number of victims; and the increase in epidemics of this disease in spite of improved hygiene and education regarding precautionary prophylaxis.

During the 1949 epidemic of poliomyelitis that swept the United States—the greatest epidemic of this disease recorded—contraction of it by definite contact with other cases was not established in an elaborate study by the New York State Department of Health. The United States Public Health Service in its studies also found the same negative answers during the same epidemic. Time Magazine,[178] commenting on these surveys, points out how, when and where people catch polio remained a mystery. Science News Letter[179] (August 19, 1950) indicates that quarantines for poliomyelitis are useless. It says: "Strict and heavy quarantines for infantile paralysis do not stop polio epidemics, health and poliomyelitis authorities agree. All attempts to stop polio by quarantine have failed and authorities now consider it foolish to enforce it."

Dissatisfaction with the virus explanation of cause of poliomyelitis appears to be evident from the fact that many theories, apposed to the virus theory, have been proposed since Landsteiner's discovery 42 years ago. Thus, there are found explanations of the cause of the disease in "globoid bodies," streptococci, poisons, allergy, vitamin deficiency, etc. Morse[180] (1929) in discussing the various etiological agents that have been described to account for this disease, i. e., filterable virus, hemolytic streptococcus, and globoid bodies, says: "They suggest the suspicion, however, that everybody may be wrong as to the etiological factor in infantile paralysis and that it may be something which has not even been thought of." This statement, made almost a quarter of a century ago, emphasizes the urgent need for unlimited research.

Some investigators have pointed out the fact that human poliomyelitis and the disease produced in experimental animals from human material, etc. are not the same disease. Toomey[181] (1935), for example, stated that intranasal and intracerebral inoculation of the poliomyelitis virus in monkeys does not produce the same disease that is seen in man.

The favorable results in the treatment of human poliomyelitis

by spinal drainage that have been noted by a number of workers[182-185] would appear to indicate: (1) benefit was derived by the draining off of a toxin, and (2) that human poliomyelitis and the experimental disease of animals are separate entities since spinal drainage in the case of the experimental animal has been found to be injurious.[186] In regard to the first of these premises, it is well-known that the poliomyelitis "virus" never has been recovered from the spinal fluid of either humans or experimental animals and that it would be impossible to drain off a virus that is fixed in the substance of the spinal cord. Therefore, the favorable results obtained in humans by spinal drainage and the unfavorable results obtained in experimental animals would appear to indicate that a poison is responsible for the human disease, whereas, a neurotropic enzyme resulting from this poisoning is responsible for the animal disease and it would be impossible to drain it off.

Toomey[191] (1939) insisted that the so-called poliomyelitis virus is an enzymatic catalytic agent, a fact that appears to reconcile the virus and the poison theories of this disease. Toomey found, for example, that the combination of bacterial or intestinal toxin and also peach skin filtrates accelerated the effect of the so-called virus when both were injected simultaneously into experimental animals.

Evidence that the human and experimental diseases are separate entities appears to be indicated by frequent observations that the heart is involved in cases of human poliomyelitis. Reporting on 35 cases of poliomyelitis in which necropsies were performed, Ludden and Edwards[192] (1949) suggest that the myocarditis in 14 cases was probably caused by the poliomyelitis virus. It was suggested that it might be possible to demonstrate the virus in such lesions and experimentally to produce them with the virus. This assumption is difficult to reconcile with the facts that the so-called virus of poliomyelitis has never been found in the blood and that it is neurotropic in action.

As further proof of the distinction between human poliomyelitis and the experimentally produced disease is the fact that drugs that have proved to be effective in the treatment of the latter have been ineffective in the former. The vaccines of the "virus" that have been prepared and proved to be effective in

controlling the experimentally produced poliomyelitis in animals, have caused in humans (as in the case of laboratory workers), the paralysis which the vaccine sought to prevent.[193]

A revision of our views regarding the virus concept of cause of poliomyelitis is called for on the basis of the fact that "viruses" can be produced in the body by poisons, and that inclusion bodies, which supposedly indicate products of virus activity or the elementary virus bodies themselves, can be found in poisoned animals. This work has been reviewed by Scobey.[194]

Although Landsteiner and others have been able to inject emulsions of spinal cord material into experimental animals to cause a paralytic disease, a number of workers have also caused paralysis in experimental animals simply by injecting normal nervous tissues.[195] This fact would indicate that some of our modern studies relating to the so-called virus of poliomyelitis require careful reappraisal. Lumsden[196] (1949) states: "Brain tissue from many mammalian species contains some non-infective chemical agent which is specifically pathogenic to the mammalian nervous system." He tested over 300 guinea pigs, 150 rats, and 12 rabbits and experimentally produced encephalomyelitis with normal brain material.

COMMENT

This report shows that there are many opportunities for poisoning with resultant anterior poliomyelitis during the seasons when epidemics of this disease are prevalent. The significant changes in fruits and vegetables, as a result of drought and lack of water; toxicity of food from bacterial contamination; chemical poisoning of fruits and vegetables during growth by fertilizers, other chemicals, and insect sprays; and chemical changes in water from decay of organic material by natural processes or bacterial decomposition with such organisms as pseudomonas or the botulinus organism have all been either ignored or not adequately correlated as factors in producing human poliomyelitis. Yet, this wide variety of poisons does produce essentially the same result, i. e., flaccid paralysis with changes in the anterior horn cells—poliomyelitis.

Putnam[197] (1888) stated, regarding poisons in general: "The very fact that such widely different agents as lead, arsenic, alco-

hol, sulphide of carbon, and the poison of the tubercular, and other bacteria induce on the whole such similar types of neuritis, suggests that there is a common factor at work which is independent of the particular agent, and must consist in some peculiarity of the affected tissues. If this is so, then a variation of the symptoms which is often met with in one of these forms of poisoning, such as that from lead, will be likely to occur to a greater or less extent in the rest, when the conditions are favorable. These peculiarities in the local accumulation of the poisonous agent must certainly affect the symptoms, even if it be true that the degree of vulnerability of the special tissues, and not the amount of the poison present, that chiefly determines whether or not paralysis will result."

The above statement indicates not only an individual susceptibility to poisons but likewise a secondary factor. It is highly possible that this secondary factor, common to most poisons, is a vitamin deficiency, presumably a multiple deficiency. This fact is suggested by the work of Hart et al.[66], Mellanby[70], and Reyes[71] which has been previously considered. There is also to be considered the sequelae of poisoning as exemplified in alcoholism with its resultant polyneuritis, paralysis, Korsikoff's syndrome and Wernike's encephalitis, for many years considered to be due to the specific effects of alcohol on the nervous system, but which are now generally recognized as caused by vitamin B_1 deficiency as secondary to alcoholism.

Post-diphtheritic and post-typhoid paralysis, formerly thought to be the direct effect of the specific bacterial toxins are now also regarded as due to a vitamin B_1 deficiency secondary to these toxins. The reports that have appeared in the literature of the development of poliomyelitis as a sequel to acute infectious diseases of childhood and secondary to pertussis inoculations are evidently of the same nature. The effectiveness of vitamin B_1 reported by some workers in the treatment of poliomyelitis would appear to indicate that a secondary vitamin B_1 deficiency does in fact exist in poliomyelitis.

One of the logical approaches in the investigation of the poliomyelitis problem would appear to be the search for a poison in the food and water of victims of this disease. Advice given in an editorial in the Boston Medical and Surgical Journal[78] over half

a century ago appears to be extremely important in emphasizing this point. It says: "One of the most significant advances of the past few years has undoubtedly been the attention paid to the importance of various toxic causes in the production of disease. The work of Van Giesen of New York and of many foreign investigators has forced upon us the conviction that the etiological problems of medicine may be much more intelligently met by widening our conception of the idea of poisons, and studying with every possible means at the disposal of our laboratories the chemical questions which such conception involves. *Unquestionably progress lies in this line of investigation.* It is self evident, as civilization advances and new products are used more and more in the arts and in our homes, that new dangers must necessarily arise, and a constantly growing list of so-called extrinsic poisons be brought into our daily life."

Truthfully, progress was being made in the investigation of poliomyelitis before the discovery of the so-called virus. Toxicologists and bacteriologists were working simultaneously on the problem, with limited funds, and apparently reporting important results. For example, it was reported from scientists in these branches of medicine that poliomyelitis could be produced both by organic and inorganic poisons as well as by bacterial toxins. However, these investigations lost support when the so-called virus theory came to be considered by some to be the full and final answer to the problem. Funds for poliomyelitis research were from then on designated only for virus studies. Another factor, and perhaps the most important one, that interrupted this pioneer work and that still stands as a barrier to unlimited research today, was the compulsory reporting of poliomyelitis as a communicable disease, which implies legally that this disease is caused by a germ or a virus.

Our forefathers, who were responsible for the fundamentals of medicine on which modern medical science is still based, incriminated food and water as the cause of diseases that not infrequently resulted in paralysis. The foundation of our knowledge of poliomyelitis was laid by observing men like Underwood, Badham, Heine, Colmer, West, Duchenene, Landry, Charcot, Erb, Gowers, Strümpel, Wickman, and many others who did not have access to the spinal puncture needle to assist them in their diagnosis

of this disease. The spinal puncture was not generally employed in the diagnosis of disease until several years after it was introduced by Quinke,[139] in 1891.

The fundamental principles of investigation of epidemics advocated by Hippocrates,[98] The Father of Medicine, included meteorology, astronomy, dietetics, physical and chemical examination of water, the effect of weather conditions on vegetation, etc. These potential factors are being ignored in the mandatory requirement of an exogenous virus as the cause of poliomyelitis. Hippocrates' statement regarding these fundamental principles of epidemic investigation: "*For from these things he must proceed to investigate everything else.*" appears to have been the mode of investigation of disease for centuries. Today under the pressure of the exclusive virus theory of cause of poliomyelitis the wisdom of Hippocrates is obscured. Dauer[1] (1938) makes the following significant statement: "The aim of epidemiology is to find out and expose, as far as humanly possible, why diseases are as they are, and to learn the rules under which they act." This statement implies, as advocated by Hippocrates over 2000 years ago, unlimited research and the taking of every possibility into consideration.

To repeat, the predominating factor that obstructs our progress in poliomyelitis investigation today and the continuance of the valuable observations made by the pioneers of medicine is obviously the "virus". This more or less general acceptance of an exogenous "virus" as the cause of poliomyelitis has obviously been influenced by subsidized exclusive "virus" research, mandatory public health laws, and continuous, widespread "virus" publicity. Poliomyelitis investigation obviously calls for a revision of exclusive concepts and for unlimited research.

SUMMARY

1. A number of theories, other than the virus theory, have been proposed to explain the epidemiology of poliomyelitis. The "poison theory", which for centuries from the time of Hippocrates has been emphasized to explain the paralytic diseases of the past and the poliomyelitis of more recent times, has been more or less generally ignored for almost half a century. Proof that poisons can cause poliomyelitis is given in this report.

2. The so-called virus of poliomyelitis is considered to be an endogenous chemical substance resulting from this poisoning. Human poliomyelitis and the experimental animal disease are regarded as separate disease entities.

3. It is pointed out that the more or less general acceptance of an exogenous virus as the cause of poliomyelitis has been influenced by exclusive virus research; mandatory public health laws that legally make poliomyelitis a communicable disease; and widespread virus publicity.

The writer wishes to express his appreciation to his wife, Alice Gilmore Scobey, R.N., for her helpful suggestions and cooperation in the preparation of this paper.

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1411 *South Salina Street.*

RESULTS OF OPERATION IN 40 CASES OF CONGENITAL HEART DISEASE. (Thorax, London, 5: 73, March 1950). Wilson reports the results of follow-up on 40 of 63 patients operated on for congenital cyanotic heart disease at the University Hospital in Copenhagen. No case was included in which less than three months had elapsed since the operation, and only 6 were included in which there had not been at least six months' observation. Thirty-five of the 40 patients had tetralogy of Fallot; 2 patients had pulmonary stenosis with interatrial septal defect; 2 had an underdeveloped right ventricle, and 1 was thought to have aortic stenosis associated with an interatrial septal defect and a small functioning patent ductus arteriosus. Two of the 5 deaths that occurred in these 40 cases must be considered as anesthetic deaths. In assessing the results of the operation, the author attempted to view each case as a whole, with attention to improvement in color, height and weight increase, absence or sign of cardiac insufficiency as well as to the important feature of increased or unchanged exercise capacity. A final analysis revealed that the result was excellent in 8, very good in 10, good in 6, fair in 8, poor in 1 and unchanged in 2.—*Journal A.M.A.*

PEDIATRICS HALF A CENTURY AGO

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

ACUTE LEUKEMIA*

Report of Case

A. D. Blackader, M.D.

Professor, Pharmacology and Therapeutics, and Lecturer on Diseases of Children, McGill University,

AND

B. D. Gillies, M.D.

Pathologist, Montreal General Hospital, Montreal.

Although numerous cases of acute leukemia in childhood have been reported, and the subject itself has recently been discussed at length by members of this Society, the affection is still sufficiently rare and obscure to demand that a careful record be made of each case. The notes of the following case, which appears to us to possess features of special interest arising from its abrupt onset and extremely rapid course, we think desirable to place on the records of this Society:

CASE REPORT

A. M., a lad of Jewish descent, aged fifteen, born in Chicago, but living in Montreal for the past three years, was admitted to the Montreal General Hospital on June 4, 1904. On May 29, seven days previously, he was awakened in the morning by a profuse epistaxis, which continued all that day and part of the next. There had been no injury and his parents could assign no cause. The day previously, he had been at school and was stated by his parents to have been in good health and his skin of a normal color. He was bright, played with his friends as usual, and went to bed, as far as they knew, in perfect health. On two or three previous occasions he had suffered from bleeding at the nose, but each of these attacks had been promptly stopped with simple remedies. Otherwise he had always been healthy, except for an attack of mumps. His diet, so far as could be learned, was wholesome.

*Read before the Seventeenth Annual Meeting of the American Pediatric Society held at Lake George, N. Y., June 19, 1905.

Reprinted from Archives of Pediatrics, 22: 890-898, 1905.

The portion of the city in which he lived is one of the most healthy, being comparatively close to the mountain. The family history is an unusually good one with no tendency to hemophilia in any of its members.

The epistaxis, which had continued all through the day of the 29th, and part of the 30th, had recurred on the 31st and had been checked by plugging the nares with gauze. On June 1 he began to bleed from the mouth, and a continuous oozing from the mucous membrane of gums and of both cheeks had persisted until the time of his admission. Ecchymotic spots had also appeared on the lower extremities. On June 2 vomiting and diarrhea had set in; the vomitus contained blood, but there had been no melena or hematuria. Headache was complained of and vertigo, with shortness of breath on any attempt at movement.

When seen in the hospital he was in a very restless state, tossing from side to side on the bed; mentally, he was dull, but could be roused to answer questions. The rectal temperature was 101° F., pulse 132 and respirations 32. Examination showed him to be a fairly well nourished boy; the skin, mucous membranes and conjunctivæ showed a marked degree of anemia; petechiæ, varying from 1 to 5 mm. in diameter, were scattered over the trunk, and more numerously on the extremities, especially on the legs below the knees where several large ecchymoses were also to be seen. There was no edema. The nares were plugged with gauze. Petechiæ were visible on the buccal surface of the cheeks and there was a continual oozing of blood from the edges of both gums. The tonsils were slightly enlarged and the breath was foul-smelling. The joints were normal. Marked tenderness was elicited by pressure over the sternum, but over no other bone. There was a moderate discrete enlargement of the superficial lymph glands, the anterior and posterior cervical, the axillary, the epitrochlear and the inguinal were easily palpable, but tenderness in them was not elicited by pressure. The respirations were slightly increased in frequency, but examination revealed no abnormal conditions in the lungs. Transverse cardiac dullness measured 7 cm.; there was a slight systolic murmur at base. Blood pressure registered 120 mm. by the Riva-Rocci apparatus.

The abdomen was distended, especially in the upper zone, and the outline of the enlarged spleen was distintely visible. On pal-

pation the lower margin of the liver was easily felt 6 cm. below the costal margin in the nipple line and tenderness was elected on pressure over it. The spleen was distinctly palpable and its resisting border could be felt 6.5 cm. from the median line and 6 cm. below the costal margin.

Hearing appeared to be normal. Vision in the right eye was blurred. An examination of the field of vision was impossible on account of the patient's condition and unfortunately no examination of the fundi was made.

The urine was somewhat turbid, its specific gravity 1.012; it was acid in reaction and contained neither albumin nor sugar. Under the microscope only a few leukocytes were to be seen, but no blood cells, no casts, and no uric acid crystals.

An examination of the blood made immediately on admission by Dr. Patterson, to whom we are also indebted for many of the notes, showed the erythrocytes, 1,620,000; leukocytes, 34,000; hemoglobin, 35 per cent.; clotting was normal.

The lad's condition became rapidly worse. Numerous loose movements with no evident signs of blood in them occurred. He vomited all nourishment taken. The petechiæ and ecchymoses increased in number and in size; vision was lost in the left eye on the morning of the sixth. Death took place the same evening.

A second blood count made on the morning of the sixth showed:

Erythrocytes	1,390,000	
Leukocytes	91,600	
Large lymphocytes	45.5	per cent.
Small lymphocytes	34.5	" "
Polymorphonuclears	12	" "
Myelocytes	3.0	" "
Transitionals	4.2	" "
Eosinophiles	.8	" "

The small lymphocytes were quite normal, the nuclei of the larger ones were perhaps more granular than usual. In a count of 500, three nucleated cells were seen. A culture from blood taken at the same time was carefully made by Dr. Gillies, but proved negative as the tubes showed no growth after forty-eight hours.

Autopsy. The autopsy was performed by Dr. Gillies sixteen

hours after death. The body was that of a well nourished boy; postmortem rigidity was present, also postmortem lividity over the dependent parts of the trunk. The skin was of pale lemon tint, showing over the abdomen, chest, legs and arms numerous petechiæ, and over the lower extremities, large purpuric patches. The external genitals were normal, the pupils equal and the gums were covered with clotted blood.

On opening the abdomen the peritoneum was seen to be glistening; the liver was seen exposed for one hand's breadth below ensiform cartilage, and the spleen reached the costal border at the level of the ninth left costal cartilage. On removing the sternum the thymus gland was seen to be enlarged.

The pleurae, lungs and pericardium were normal. The *heart* was slightly enlarged, and its muscle pale. Lying beneath the visceral pericardium, on the anterior surface of the ventricles, close to the septum, were seen numerous fairly large ecchymoses. The *spleen* was large, its weight 430 grams; its capsule was slightly wrinkled; its consistency increased. On section, the organ cut firmly and presented a somewhat mottled color, varying from a dark brownish-red to a dark yellow; the malpighian bodies showed distinctly on the dark background. The *right kidney* was of normal size; its weight 100 grams; its color pale; the venæ stellatæ were prominent. The capsule peeled off readily from the surface of the organ, showing several whitish areas, not raised above the level of the surrounding kidney substance, and of varying size. On section, the cortex and medulla were seen to be of a pale yellowish color, and scattered over the cut surface were irregular whitish masses, similar to those seen on the outer surface of the organ. The pelvis was normal. The *left kidney* presented the same general appearance as the right, but the whitish masses were fewer in number. The *suprarenals* were normal. The *liver* was slightly enlarged, its color brownish-yellow, its weight 1,330 grams, its capsule smooth, its edges sharp and its consistence normal. On section the color was yellowish and the lobules somewhat indistinct; the parenchyma was friable. Scattered over the cut surface were seen yellowish-white masses which were less distinct and smaller than those in the kidneys.

The stomach was somewhat dilated and showed submucous

ecchymoses. In the duodenum, immediately beyond the pylorus, was seen a localized infiltration of the walls resembling a tumor 3 cm. in breadth. The surface was elevated and the mucous membrane eroded. The lymph follicles throughout the small intestine showed hyperplasia and the Peyer's patches were much enlarged. The colon was normal. The pancreas was normal. Behind the pancreas was a large mass of swollen lymph nodes, of firm consistence, one node being nearly as large as a small lemon. The edge of the mass extended as far as the site of the eroded area in the duodenum. Some of the nodes in the mass were of a reddish hue, while others were whitish and firm. The mesenteric lymph nodes were swollen and soft, as also were the inguinal, the axillary and the cervical nodes. The rectum was normal. The testicles were normal. The thymus was enlarged, soft and gelatinous on section; the thyroid colloid. The larynx and trachea were normal. The esophagus was normal. The abdominal aorta was smooth and elastic.

Microscopical Appearances. The duodenum, in a specimen taken from the tumor-like swelling immediately beyond the pylorus, showed an infiltration of large round mononuclear cells into the mucosa and submucosa between Brunner's glands. The cells were not granular and among them might be seen a few small mononuclear cells resembling small lymphocytes. The normal tissues of the gut in this section seemed little disturbed by the presence of these cells. A section at the level of the tumor, however, presented a different microscopical picture. Here all signs of the normal structures were lost, except in the neighborhood of the outer edge of the tumor where a few strands of muscular fibres were seen passing in among masses of mononuclear cells. The cells presented no definite structural arrangement; in places a reticulum could be made out about each cell, but in other places the reticulum was lost and the cells were massed together and held in place by a coarser mesh. The blood vessels were poorly developed. The microscopical appearance resembled that of a lymphosarcoma. The mucosa was completely absent from the surface of the tumor.

The capsule of the spleen was seen to be somewhat thickened, the parenchyma everywhere showed marked hyperemia, the sinuses being distended with blood cells. The malpighian bodies

were enlarged and showed a general infiltration with mononuclear cells of a uniform type, in which the nucleus was relatively large and the protoplasm clear.

The liver cells in general stained well and the nuclei were clear and distinct, but in many there were evidences of fatty change. In many places about the portal vessels were to be seen collections of mononuclear cells; masses of these cells were also scattered irregularly throughout the lobules, leading in some places to the complete disappearance of the liver cells. It was possible to trace in places the various stages from that of slight compression of the liver cell to that of complete replacement, the stage depending upon the number of newly formed mononuclear cells. In some instances atrophying liver cells could be seen within a mass of these round mononuclear cells.

The kidney showed the cells of the glomeruli distinct, with clear staining nuclei. In the convoluted tubules the cells were somewhat swollen and the nuclei indistinct, but in other parts the cells stained clearly. Throughout the section masses of well stained mononuclear cells were to be seen forming the whitish masses visible to the eye on section. In some places these compressed the normal tissues about which they lay, and in other places replaced them completely. Where the new-formed cells were few in number no change could be detected in the pre-existing structures.

The mesenteric, retroperitoneal, cervical and axillary lymph nodes all presented the same microscopical appearance. Among the ordinary cells of the lymph nodes were to be seen numerous large mononuclear, non-granular cells. In some of the retroperitoneal nodes polynuclear cells were also present. The structure of the lymph nodes was preserved.

The bone marrow showed myelocytes, both neutrophile and eosinophile; also red blood corpuscles, both nucleated and non-nucleated. The large mononuclear lymphocyte predominated all other white cells, and in comparison to it all the other white cells were relatively few in number.

This case, both in its clinical symptoms and its anatomical findings, is, we think, to be regarded as a typical example of acute lymphocytic leukemia, as recognized at present. It began, as many of these cases do, abruptly with hemorrhagic manifesta-

tions. Close cross-questioning of both parents and the lad himself revealed no indication of ill-health in the boy previous to the initial epistaxis, except that in answer to a leading question the lad said that after running he had perhaps felt a little out of breath for the past three weeks. It had not, however, interfered with his games. Counting from this initial epistaxis the course of the disease was just ten and a half days. The case in this respect is therefore especially interesting, for so far as we have been able to investigate the subject, the cases which have ended fatally in twelve days, or less, may be counted on the fingers of one hand. Litten would seem to hold the record, his case lasting only four and a half days. Nobel reports one the duration of which was ten days. Greiwe's case lasted eleven days, and those of Englisch and Obrastzow lasted twelve. McCrae also reports a case in a lad of fifteen years, which ran its course in about twelve days.

The question is an interesting one as to what the exact condition of the lymph nodes and blood in this lad was immediately preceding the hemorrhage. While there was nothing in his appearance or in his behavior to attract the attention of his parents who were rather above the average of their class in intelligence, we recognize that in a certain number of cases a latent stage commonly known as the aleukemic stage exists, and it is possible that a careful examination at that time might have revealed some deviation from the normal.

Dr. Thos. McCrae, in speaking of 5 cases that occurred in the Johns Hopkins Hospital, gives as the average of their blood count on admission: Hemoglobin, 35.4 per cent, red cells 1,822,000 and leukocytes, 104,000. In the case we have just reported the hemoglobin was 35 per cent., the red cells 1,620,000, and the leukocytes 34,000 (1—48); but in thirty-six hours the leukocytes had risen to 91,000 (1—15); at the same time the large lymphocytes were greatly increased, the polymorphonuclears were diminished and myelocytes were present.

Dr. McCrae considers that in acute leukemia we have to recognize in addition to what may be called the specific leukemic features, grave changes in the red cells themselves, suggesting that in acute leukemia, in addition to the changes in the white cell count, we have to deal with a very rapid and severe anemia of

the primary type. Nevertheless, he admits that in the majority of cases, and especially in rapid cases, there is very slight variation in the red cells in size, shape and staining. In the present case there was almost no poikilocytosis and no polychromatophilia. A careful recount showed a few nucleated red cells with no mast cells; the character of the staining was extremely uniform.

According to Pinkus, of Berlin, children are attacked with relative frequency. This must hold true of the second rather than of the first decade. Of 45 cases recorded by Theodor, 6 occurred in the first decade and 5 between the ages of ten and fifteen years; and in 56 cases collected by Fussell, Jopson and Taylor, 9 were in the first decade and 5 between ten and fifteen years. In the 5 cases reported by McCrae, one was aged three and one fifteen. Nevertheless, Churchill, in his paper read before the Society last year, was only able to collect 20 cases under ten years of age reported in medical literature.

On the relative frequency of acute leukemia, statistics, beyond indicating its comparative infrequency, are not very definite. McCrae states that in 17,100 admissions into the medical wards of the Johns Hopkins Hospital, there were thirty-seven with leukemia, and of these only five were acute. In Montreal, out of 9,930 medical admissions into the Royal Victoria Hospital, there were 21 cases of leukemia, of which 5 were regarded as acute, and in 10,350 admissions to the medical wards of the Montreal General Hospital, there were 8 cases of leukemia, of which only one was acute.

Acute leukemia in childhood differs in few respects from the disease as met with in adult life. Hutchison, following Guinon and Joly, describes three types of the disease. In the first there is a profound anemia with general glandular enlargement, but hemorrhages appear only in the latter stages of the disease. In the second type the hemorrhagic diathesis is manifested from the outset, petechiæ appear early and the case may assume the aspects of an infective purpura. In the third type, lesions of the buccal cavity are the most striking features.

While the bone marrow is more or less affected in every case, cases are reported in which no splenic or glandular enlargement is present. Kelly has called attention to the fact that while the diagnosis is to be made from the blood examination alone, a single

enumeration is not always sufficient. In one of Kelly's cases the leukocytic count showed only 5,200 leukocytes, yet the differential count showed over 90 per cent. of lymphocytes.

Between acute leukemia and chronic leukemia there is no definite dividing line, and although certain authorities have regarded the two affections as different entities, the clinical symptoms and blood examinations present no essential point of difference between the rapidly malignant and the more indolent forms of the disease.

That there may be a close relationship between leukemia and pseudo-leukemia would appear very probable. In an interesting paper published by Dr. Martin in 1896, attention was called to this fact, and to the tendency to fall back from the present somewhat sharp classification of these diseases and to return to the simple grouping of Trousseau, who included under the single term *adenie* all lymphoid hyperplasias.

In a recent paper Turck states that he would include under the term lymphomatosis all true hyperplastic tumor formation of lymphoid tissue due to formative disturbances. Thus into one group with this appelation he places all cases of lymphatic leukemia, acute leukemia, pseudo-leukemia, lymphosarcoma, lymphosarcomatosis, chloroma, malignant lymphoma and, perhaps, also, splenic anemia. The fundamental basis of the group he regards as an increased stimulation of growth in the lymphatic system. The etiology is unknown. The difference in the manifestations depends upon two factors:

1. The degree of the increased growth and its local behavior.

2. The presence or absence of a washing out of cells, the result of this growth, into the peripheral blood stream. In one subgroup all the new cells go to the formation of tumor masses and the blood picture is unchanged. The blood finding is alymphemic. In a second and larger sub-group, besides the cells making up the tumor masses, there is an increased washing out into the blood of the cells representing the product of this increased growth, the lymphocytes.

Turck does not deny that the bone marrow may be involved, and even admits the possibility of this being occasionally the sole site of the disease, but he does deny the absolute dependence of the lymphemia upon the blood marrow changes in all cases.

BOOK REVIEWS

MITCHELL-NELSON TEXTBOOK OF PEDIATRICS. Edited by Waldo E. Nelson, M.D., with the collaboration of 63 Contributors. 5th Ed. Cloth. Pp. 1658. Illustrated. Price $12.50. Philadelphia: W. B. Saunders Co., 1950.

This book can be highly recommended both as a teaching text and as a reference volume. The book covers the field of pediatrics adequately and the format of the book makes for easy reading. It is extremely difficult to review a book of this size. However, a sampling in the form of excerpts might give an indication of its value. In one of the early chapters one finds the following regarding the thymus: "Today it is recognized that there are many reasons for respiratory difficulty in the newborn period. The thymus has never been directly implicated as a cause of this difficulty and it is fortunate that such radiation, with its possible hazards, is becoming a rare procedure." The mental and emotional development are succinctly presented and it's worth quoting what Plant and Senn have to say about "punishment": "Punishment should be kept at a minimum. It should be short and meaningful when administered, and should be related in time as closely as possible to the wrongdoing. In some instances, spanking may be the only punishment which fits the specifications; for other children, deprivation may be more effective; and many children are adequately disciplined merely by words. So far as possible children should know what act will incur a penalty. Corporal punishment has fallen into disrepute because many parents use it to vent their own exasperation; but, properly administered, a spanking is much to be preferred to lengthy and vague verbal attempts to clear the situation."

Feeding problems in infants and children are protean in their manifestations. Of the many means of treating them Nelson cautions that the routine use of sedatives, as elixir of phenobarbital, is to be deplored; it would appear that the practice is extensive. The problems of the upper respiratory tract are at times complex and defy unanimity of opinion. Nelson is justifiably conservative about the most common problems of childhood. In regard to the tonsils, he states that there is no certain way to demonstrate by

direct observation whether tonsils are or are not harboring chronic infection of an order which would justify their removal. Every attempt should be made, however, to postpone the operation until the child is at least four or five years of age and it should not be performed as a prophylaxis against the common cold at any age. It should be noted that it has been claimed that the incidence of sinusitis is greater in children who have had their tonsils and adenoids removed. The book has many fine illustrations and pictures, and the appendix at the back of the book has many tables which are valuable as an easy reference source.

MICHAEL A. BRESCIA, M.D.

LANGUAGE FOR THE PRESCHOOL DEAF CHILD. By Grace H. Lassman. Cloth. Pp. 263. Illustrated. Price $5.50. New York: Grune & Stratton, 1950.

The deaf child can learn to talk. However, teaching the child to talk is a problem which requires the cooperation of all individuals who come in contact with the child—the teachers, the parents and the neighbors who make up the child's environment. The author shows the interrelationships of the different groups and how best to approach the handicapped child. The young deaf child must be given time to realize that he has a voice which he may use for a purpose. In guiding the young deaf child, two principles must be adhered to by parent and teacher: 1. His needs as a child must be provided for, by exposure to a normal, happy environment in which he may enjoy the activities and experiences of the hearing child. 2. His needs as a deaf child must be met, by special, consciously designed methods employed by the adults who guide him. It has been shown that many emotional problems of the deaf adolescent and adult, socially, academically, and vocationally, have their roots in their parents' attitudes toward the handicap. A course in the training of the preschool deaf child, which will be sent free of charge to any parent of a deaf child under five or six years of age, anywhere in the world, can be obtained by writing to John Tracy Clinic, 924 W. 37th St., Los Angeles 7, Calif. This book is highly recommended to physicians and to teachers who may deal with the deaf child. MICHAEL A. BRESCIA, M.D.

ITEMS

Effect of Alcohol on Development of Children. (Archiv für Kinderheilkunde, Stuttgart, 138:199, 1950). Fischer made studies on children in a region where grape culture and wine production represent the chief source of income. The majority of the children were the offspring of families who had lived in this wine growing region for generations. The wine growers consume daily an average of 3 to 5 liters of so-called "house drink" (second-rate wines) with an alcohol content of 7.24 per cent by volume. The women drink less, but still considerable amounts, and the children frequently get their first drink of wine on the first or second day of life and later are often given wine in their milk to quiet them. The author made studies on 1,938 school children of both sexes. Comparing observations on the children of the wine growing villages with those on the city children revealed that chronic consumption of alcohol by parents and children apparently did not have a deleterious effect on the development of the children. On the contrary, the children in the wine growing rural regions showed a better physical development.—*Journal A.M.A.*

Familial Distribution of Diabetes Mellitus. (Annals of Eugenics, London, 15:95, March 1950). Harris found in a study of 1,241 diabetic patients that 4.34 per cent of the sibs of these patients were also diabetic. The sibs of the patients in whom the disease commenced in early life are more likely to develop the disease in early life than are the sibs of the propositi in whom the disease developed in later life. About 7 per cent of the sibs of propositi with age at the onset below 30 years may be expected to develop the disorder by the age of 40, while only about 1.3 per cent of the sibs of propositi who developed the disease after 30 years may be expected to develop it by the age of 40. Five and three hundreds per cent of the parents of the propositi had diabetes. There appeared to be little or no parent-child correlation with respect to age at onset of the disease. Less than 1 per cent of the children of the propositi were found to be diabetic. It was estimated that about 1.4 per cent of children of a diabetic parent may be expected to develop the disease by the age of 40.—*Journal A.M.A.*

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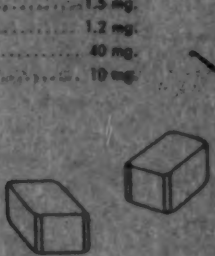

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